Clinical Case Report

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Delayed surgery in a patient with pneumococcal peritonitis and bacteremia secondary to perforation of gastroduodenal ulcer

A case report

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Abstract

Rationale: Very few cases of secondary peritonitis caused by Streptococcus pneumoniae have been described in the literature, and they have been found to occur mostly in patients with predisposing factors. Here, we report the case of an elderly patient who developed pneumococcal peritonitis secondary to perforation of gastroduodenal ulcer.

Patient concerns: An 82-year-old man was admitted to intensive care unit (ICU) for septic shock with cardiac impairment 1 day after arriving in the Emergency Department.

Diagnoses: The patient presented with pneumococcal bacteremia and pneumococcal antigenuria. No abdominal defense was found on examination. A computed tomography scan revealed pneumoperitoneum and peritoneal effusions.

Interventions: The patient was treated with effective empiric antibiotic therapy, and delayed surgery.

Outcomes: The patient gradually improved and was discharged from ICU on day 14. The ultimate outcome was unfavorable, with death occurring on day 28.

Lessons: This rare infection can occur in elderly patients even in the absence of other predisposing factors. Secondary peritonitis may be suspected in patients with positive pneumococcal antigenuria or unexplained pneumococcal bacteremia, especially if an asthenic form is possible.

Abbreviations: ICU = intensive care unit, MIC = minimum inhibitory concentration.

Keywords: bacteremia, peritonitis, *Streptococcus pneumoniae*

1. Introduction

Streptococcus pneumoniae is usually responsible for spontaneous bacterial peritonitis in patients with cirrhosis or nephrotic syndrome, or in young healthy children.[1–3] It is rarely implicated in peritonitis secondary to organ perforation. Nevertheless, a few cases of secondary pneumococcal peritonitis have been reported, including in young women with an intrauterine device, in women after delivery, and in patients (especially children) with complicated appendicitis.[4–8] In addition, 2 studies have reported 5 cases of community peritonitis secondary to perforation of gastroduodenal ulcer in patients with predisposing factors.[8,9] Here, we report a misleading case of fatal peritonitis with pneumococcal bacteremia secondary to perforation of gastroduodenal ulcer which occurred in an octogenarian patient. The patient being deceased, informed consent for this publication was given by his wife. Ethical approval was not required given the methodology used.

2. Case report

An 82-year-old man came to the Emergency Department after suffering for several days from epigastric discomfort triggered by physical effort. The patient had a history of high blood pressure, atrial flutter, nondialyzed chronic renal failure, and benign prostatic hypertrophy. He had been weaned from alcohol 20 years earlier. On admission, his treatment included losartan, hydrochlorothiazide, acetylsalicylic acid, and *Serenoa repens* extract. Clinical examination revealed a blood pressure of 115/70 mm Hg, a heart rate of 70 beats per minute, a normal temperature, tachypnea, and a bladder globe. The biology showed hepatic cytolysis 20 times the norm, a V factor of 22%, and lactate acidosis (5 mmol/L). Blood levels of paracetamol were low (6.9 mg/L). Abdominal ultrasonography showed normal hepatic parenchyma without abnormality of the bile ducts, right pleural effusions, and a bladder globe with bilateral hydronephrosis.

Treatment with N-acetylcysteine was immediately initiated. The next day, the patient worsened clinically, and he presented

Editor: N/A.

The authors have no conflicts of interest to disclose.

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Medicine (2018) 97:28(e11323)

Received: 8 March 2018 / Accepted: 5 June 2018

http://dx.doi.org/10.1097/MD.0000000000011323

with persisting hyperlactatemia of 4.7 mmol/L. Echocardiography showed a dilated left ventricle with an estimated ejection fraction of 20% and low cardiac output, prompting the initiation of dobutamine treatment. Coronary angiography was normal. The patient then developed fever and chills, which prompted investigation and that led to the introduction of antibiotic therapy with piperacillin and gentamicin.

In view of these developments, the patient was transferred to intensive care unit (ICU) on day 2. Clinical examination revealed apyrexia, satisfactory hemodynamic status, and epigastric pain without abdominal defense. A thoraco-abdominopelvic computed tomography scan (CT-scan) showed normal liver and spleen, pneumoperitoneum, and peritoneal and pleural effusions with no evidence of mesenteric ischemia, highlighting the presence of secondary peritonitis. In addition to symptomatic management of organ failure (including administration of dobutamine and norepinephrine, mechanical ventilation, and renal replacement therapy), antibiotic treatment with piperacillin-tazobactam, gentamicin, and fluconazole was conducted. Laparotomy was performed on the same day, revealing a perforation of the first duodenum with peritoneal effusions (1500 mL). The anatomopathological examination of operative biopsies concluded in a perforated ulcer.

Microbiological analysis was performed to identify the germ causing the infection. Pneumococcal antigenuria was positive. Blood cultures on admission to ICU became positive for *S pneumoniae* serotype 3 after 8 hours of incubation. Peritoneal fluid culture was positive for *S pneumoniae*, *Klebsiella pneumoniae*, *Staphylococcus aureus,* and *Candida albicans*. Alpha-hemolytic colonies of *S pneumoniae* on CNA Agar (Biomerieux, Marcy l'étoile, France) were identified using a sensitive OPTO-F optochin disk (Biomerieux, Marcy l'étoile, France) and a dryspot agglutination test (Oxoid, Basingstoke, UK). The antibiogram was performed on MHF with Biorad discs (Biorad, Marne la coquette, France). The strain showed a wild-type profile intermediate with ofloxacin, as defined by the European Committee on Antimicrobial Susceptibility Testing (EUCAST 2016). Minimum inhibitory concentrations (MICs) were measured using the E-test method (Biomerieux, Marcy l'étoile, France): Penicillin G. levels were 0.023 mg/L, amoxicillin levels were 0.023 mg/L, ceftriaxone levels were 0.016 mg/L, and cefotaxime levels were 0.012 mg/L. Typing was performed by the National Reference Center using the slide latex agglutination method. These results confirmed the diagnosis of pneumococcal peritonitis secondary to perforation of gastroduodenal ulcer.

Additional examinations were performed in the context of this pneumococcal bacteremia. Transesophageal echocardiography did not suggest endocarditis. The patient was not known to be cirrhotic, and the abdominal ultrasound performed showed normal liver parenchyma and vascularization, later confirmed by CT-scan. Plasma protein electrophoresis revealed a nonspecific inflammatory profile with hypogammaglobulinemia of 5.6 g/L. HIV and HCV serology were negative and HBV serology suggested immunity. Lumbar puncture was not performed given the absence of neurological signs. No other infectious foci (including otitis) were found.

In view of the above, antibiotic therapy was modified on day 4 to amoxicillin-clavulanic acid (2–4 g per day according to renal function) and fluconazole (400 mg per day) for a total duration of 7 days. As the patient gradually improved, he was weaned from dobutamine on day 3 and from norepinephrine on day 6. Renal replacement therapy was performed on day 6, and the patient was weaned from mechanical ventilation on day 8. There persisted biventricular dilatation and a left ventricular ejection fraction of 20%.

While the outcome was initially favorable, the patient worsened after a few days. The presence of a suspicious liquid in an abdominal drain suggested nosocomial peritonitis, and antibiotic therapy was modified accordingly (to imipenem 3 g per day). However, additional examinations did not confirm this hypothesis. The patient eventually improved, and he was discharged from ICU on day 14. An episode of cardiac failure with thrombosis of the right atrium and right iliac vein resulted in patient death on day 28.

3. Discussion

We thus report a case of peritonitis with pneumococcal bacteremia secondary to perforation of gastroduodenal ulcer. Studies have found that the incidence of perforation of gastroduodenal ulcer ranges from 7 to 10 cases per 100,000 persons and complicates less than 11% of gastroduodenal ulcers.[9,10] The 2 most commonly performed examinations for the diagnosis of intra-abdominal infection are ultrasonography and CT-scan. While CT-scan has higher sensitivity and specificity than ultrasonography, concerns about radiation exposure have recently prompted reappraisal of the roles of ultrasonography.[11] However, in the case of our patient, the secondary peritonitis could not be diagnosed with abdominal ultrasonography, but only with the CT-scan. This unfortunately caused a delay in surgery.

Three different bacteria were found in the patient's abdomen, but only *S pneumonia* was detected in his blood. Moreover, other than age, the patient had no predisposing factor to infection with *S pneumoniae*. Given the absence of other infectious foci in the patient, we can conclude that *S pneumonia* played a predominant pathological role in the peritonitis.

While management of perforated gastroduodenal ulcer is usually surgical, it may be conservative (nonoperative) under certain conditions.[12] In the case of our patient, a conservative, nonoperative strategy could not be implemented due to the presence of hemodynamic instability.[12] Unfortunately, delayed surgery after failure of conservative treatment in cases of perforated gastroduodenal ulcer is associated with a high mortality rate of around 50%.[13] By contrast, antibiotic treatment of pneumococcal peritonitis does not pose any particular problem. Indeed, a study of primary pneumococcal peritonitis has found no link between mortality and the resistance of *S pneumoniae* to penicillin.[14]

Very few cases of pneumococcal peritonitis secondary to perforation of gastroduodenal ulcer have been reported in the literature. Rueda et al[15] reported a series of 136 cases of pneumococcal infection, which included 5 cases of primary peritonitis (3.7%) and no case of secondary peritonitis. The main pathophysiological hypothesis put forward to explain these is direct colonization from the oropharyngeal tract, which can be favored by low gastric acidity.[8] The advanced age of our patient seems to confirm this hypothesis.

4. Conclusions

Our case study confirms the existence of pneumococcal peritonitis secondary to perforation of gastroduodenal ulcer. This rare infection can occur in elderly patients even in the absence of other predisposing factors. Secondary peritonitis may be suspected in patients with positive pneumococcal antigenuria or unexplained pneumococcal bacteremia, especially if an asthenic form is possible.

Author contributions

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